tion. The best response rate was 21.4% partial response (PR) and 29% stable disease (SD) according to RECIST (disease control rate 50%). With a median follow-up time of 6.1 months, the six-month survival rate was 77.5%,and the median time to progression was 84 days.76% of patients had improvement in symptoms after taking erlotinib.

Conclusion: Erlotinib demonstrated clinically significant antitumor activity and favourable tolerability as monotherapy in this series of Chinese patients with advanced NSCLC and was also associated with remarkable symptom relief .

P3-140 NSCLC: Molecular Targeted Therapy Posters, Wed, Sept 5 – Thurs, Sept 6

Erlotinib treatment after progression on gefitinib in non-small cell lung cancer(NSCLC)

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Background: Epidermal growth factor receptor has been extensively studied as a target of therapy in the NSCLC. Gefitinib and erlotinib are the tyrosine kinase inhibitors (TKI's) of EFGR. The two drugs show similar chemical structure and mechanism but differ in phamatokinetics (higher AUC in erlotinib). Despite early dramatic and durable responses in some patients, the majority of these patients eventually develop disease progression after initial response to these agents. The activity of erlotinib in patients who had disease progression following gefitinib treatment is unknown.

Material and Methods: Patients with NSCLC who had treatment with erlotinib (150mg/day) after progression on gefitinib (250mg/daily) were analyzed.

Result: A total of 19 patients who had advanced or recurred NSCLC, 14 patients were evaluable for response but 5 patients did not have an evaluable lesion or sufficient treatment duration for response evaluation (due to adverse effect or follow up loss). 12 patients were female and 2 were male. Patients' age ranged from 34 to 76 (median 53). 1 patient was a smoker, the others were a never-smoker. The histologic type was adenocarcinoma (n=13), bronchioloalveolar carcinoma (n=1). Prior best overall response to gefitinib was PR 42%, SD 29% and PD 29%. 12 patients were treated with erlotinib as a 3rd or 4th line therapy after gefitinib and the others were treated as from 6th to 7th line chemotherapy. One patient showed a partial response (7%), another patient stable disease (7%), and the others disease progression (86%). The patients with PR or SD were female and never-smoker. The prior response to gefitinib was SD in these two patients. The pathologic type was adenocarcinoma in patient with PR, and bronchioloalveolar carcinoma in patient with SD. According to prior overall response to gefitinib, median time to progression of erlotinib was 0.8 months (95% CI 0.64-0.96) in PD, 0.93 months (95% CI 0.73-1.13) in PR and 2.1 months (95% CI 0.69-3.51) in SD (p=0.14).

Conclusion: This result suggests that the activity of erlotinib after progression on gefitinib is limited. Further investigation for mechanism of resistance and 2nd response to TKI is needed.

P3-141 NSCLC: Molecular Targeted Therapy Posters, Wed, Sept 5 – Thurs, Sept 6

Assessments of the epidermal growth factor receptor mutations and amplification and their clinical implication in patients with lung adenocarcinoma

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Background: Although the epidermal growth factor receptor (EGFR) has emerged as a promising target for patients with EGFR overexpressing non-small cell lung cancer, the impetus to standardize for their prognostic and predictive value is still much needed. In this study, we performed a study to establish a relationship among EGFR mutation, EGFR gene copy, and responses for gefitinib therapy in patients with lung adenocarcinoma.

Methods: The EGFR status was evaluated by direct DNA sequencing of exons 18, 19, and 21 and fluorescence in situ hybridization (FISH) in 284 patients, and the clinical data including gefitinib treatment were gathered.

Results: Among 284 biopsy tumor samples, which were more than 0.1 cm, EGFR mutation with lung adenocarcinoma, mutation analysis was possible in 263 cases (92.4%) and gene amplification analysis was possible in 270 cases (95.1%). Male:female ratio was 44.4%:55.6% and median age was 58 (range, 21-84). One hundred and eighteen patients (44.9%) had EGFR mutations and 30 patients (11.1%) had EGFR gene amplifications. The mutations included deletion or mutation of exon 19 in 65 patients (55.1%), mutation of exon 21 in 43 patients (36.4%) and mutation of exon 18 in 10 patients (8.5%). Of 172 non-smoking patients, 92 patients (53.5%) had EGFR mutations in contrast to 26 of 91 (28.6%) in smoking patients. Of 177 non-smoking patients, 24 patients (13.6%) had EGFR gene amplification in contrast to 6 of 93 (6.5%) in smoking patients. Of 30 patients with EGFR gene amplification, 27 patients (90%) harbored EGFR mutation simultaneously. One hundred and twenty-two patients were treated with gefitinib and 84 patients had measurable lesion. In the patients with measurable lesion, response rate in patients with EGFR mutation was 69.7% (23 of 33 patients), in contrast to 24.4% (10 of 41 patients) in patients without mutation. The response rate in the patients with EGFR gene amplification was 83.3% (5 of 6 patients). Of the patients treated with gefitinib, fifty eight patients with EGFR mutation had significantly prolonged time to progression (TTP) than 50 patients without mutation (median survival, 13.1 vs. 2.6 months; p = .0003). Of 58 patients with EGFR mutation, 13 patients with simultaneous EGFR gene amplification showed much longer TTP (median survival time not reached).

Conclusions: EGFR mutation and EGFR gene amplification could be tested with small sized biopsy sample. We showed its clinical importance with regard to gefitinib. Lung adenocarcinoma that harbored EGFR mutation and gene amplification simultaneously showed better tumor response and TTP with gefitinib.